SHORT REPORT

Popliteal Venous Aneurysm Causing Pulmonary Embolism and Paradoxical Embolisation in a Patient with Antiphospholipid Syndrome

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Abstract

Introduction: Popliteal venous aneurysms are rare and can cause recurrent pulmonary emboli despite adequate anticoagulation.

Report: A 43-year-old patient with known antiphospholipid syndrome developed an extensive pulmonary embolus and ischaemic stroke despite anticoagulation. Duplex ultrasound confirmed a right popliteal venous aneurysm containing non-adherent multi-layered thrombus. At operation an 8 cm × 5 cm true aneurysm of the popliteal vein was excised. A postoperative echocardiogram revealed a patent foramen ovale.

Discussion: This case is unusual as the patient suffered a paradoxical embolism due to his patent foramen ovale. Although antiphospholipid syndrome is associated with venous thrombosis, this is usually prevented by therapeutic anticoagulation.

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Introduction

Venous aneurysms are uncommon but popliteal venous aneurysms (PVAs) are rare, with less than 120 documented cases. The incidence of PVAs is unknown as most are small and asymptomatic. Although the majority of documented cases report a strong association with pulmonary emboli (PE) (70−80% of cases), other presentations include a swelling in the popliteal fossa and post-thrombolytic syndrome. Only one previous case of a PVA associated with a stroke has been reported, where an 18-year-old woman suffered ischaemia to her left basal ganglia secondary to a 3.5 cm × 4 cm PVA and patent foramen ovale. The patient did not have a PE. The defect was repaired surgically, as was the PVA, but no thrombus was noted within the PVA.

Report

A 43-year-old man on lifelong warfarin therapy for known antiphospholipid syndrome (lupus anticoagulant positive and weakly positive for anticardiolipin) and previous...
axillary vein thrombosis, deep vein thromboses and multiple pulmonary emboli, presented with rectal bleeding following a stapled removal resection of rectum 11 days previously. His anticoagulation had been switched to intravenous heparin during his perioperative period, before restarting warfarin. The patient was resuscitated, and his international normalised ratio (INR) of 11 and anaemia of 6 g/dL corrected. The bleeding per rectum stopped and he was again recommenced on intravenous heparin. Despite this anticoagulation he developed an extensive PE and, two days later, a left parietal lobe embolic stroke.

Initially the PE was thought to be due to normalisation of his INR with inadequate heparinisation, but the occurrence of the subsequent stroke required further consideration. A non-pulsatile soft swelling in his right popliteal fossa was thought to be the origin for these embolic events, because duplex ultrasound confirmed a large PVA, containing non-adherent multi-layered thrombus (Fig. 1). As anticoagulation did not prevent systemic emboli, further interventional therapy was indicated. Although a vena caval filter might have prevented further emboli, surgery was preferred to remove the source of thrombus generation. An 8 cm × 5 cm true aneurysm of the popliteal vein (with thrombus confined to the aneurysmal sac) was excised and continuity restored with a contralateral long saphenous vein graft (Fig. 2). A patent foramen ovale was seen on subsequent echocardiography, which is being considered for closure. Postoperatively the patient made a slow recovery and has had no further embolic events.

Discussion

A venous aneurysm is a solitary area of venous dilatation that occurs in a non-varicose vein that is not associated with a pseudoaneurysm or arteriovenous communication.4 The popliteal vein measures 5–7 mm in diameter and is considered aneurysmal when it is larger than 20 mm.1–4 Exact aetiology is unknown; trauma, inflammation, congenital weakness (e.g. elastin insufficiency in the vessel wall) and localized degenerative change have been suggested.1,2

Venous hypertension is not thought to be relevant, as most aneurysms develop in low-pressure systems (lower limb and neck).4

Antiphospholipid syndrome is characterised by recurrent venous or arterial thromboses.5 Thrombus formation is thought to be due to activation of vascular endothelium (facilitating platelet adhesion) and antibody reaction to oxidized low-density lipoproteins, predisposing to atherosclerosis and myocardial infarction.5 The risk of recurrent thromboembolic events in patients with antiphospholipid syndrome is 10%, which decreases to 1–2% with anticoagulation (INR 2–3).5 Patients positive for both lupus and anticardiolipin anticoagulant are at an increased risk of thrombus formation.5

PVAs can be detected by duplex scanning, computed tomography, magnetic resonance imaging or venography.1,4 Treatment depends upon the size and symptoms associated with the PVA.2 Small aneurysms are thought to carry a low risk of emboli and can be managed by anticoagulation and duplex surveillance.2 Large aneurysms are associated with an 80% chance of PE formation despite anticoagulation.2 Surgical excision of the aneurysm is advocated as the mainstay of treatment for these larger aneurysms (with or without venous reconstruction), and PE recurrence postoperatively has not been documented.5 The use of a vena caval filter in the presence of a PVA has not been reported.

This case illustrates the need to investigate for a cause of PE and the surgical intervention required to treat symptomatic PVAs, when such patients are symptomatic despite anticoagulation. Our case is unusual due to several factors. First, the patient has a PVA; second, he had a PE and stroke despite full anticoagulation; third, he had a patent foramen ovale and fourth, he had antiphospholipid syndrome. Although both of the embolic events sustained by this patient could have been due to the antiphospholipid syndrome, the fact that they occurred while he was fully anticoagulated makes it more likely that they came from his PVA. It is therefore likely that this patient’s stroke was due to an embolus passing across his foramen ovale.
References


